1. Muscular System Module 1: Overview of Muscle Tissues
2. Muscular System Module 2: Skeletal Muscle
3. Muscular System Module 3: Contraction and Relaxation
4. Muscular System Module 4: Nervous System Control of Muscle Tension
5. Muscular System Module 5: Exercise and Muscle Performance
6. Muscular System Module 6: Cardiac Muscle Tissue
7. Muscular System Module 7: Smooth Muscle
8. Muscular System Module 8: Gross Anatomy of the Muscular System

Muscular System Module 1: Overview of Muscle Tissues
By the end of this section, you will be able to:

- Describe the different types of muscle
- Explain contractibility and extensibility

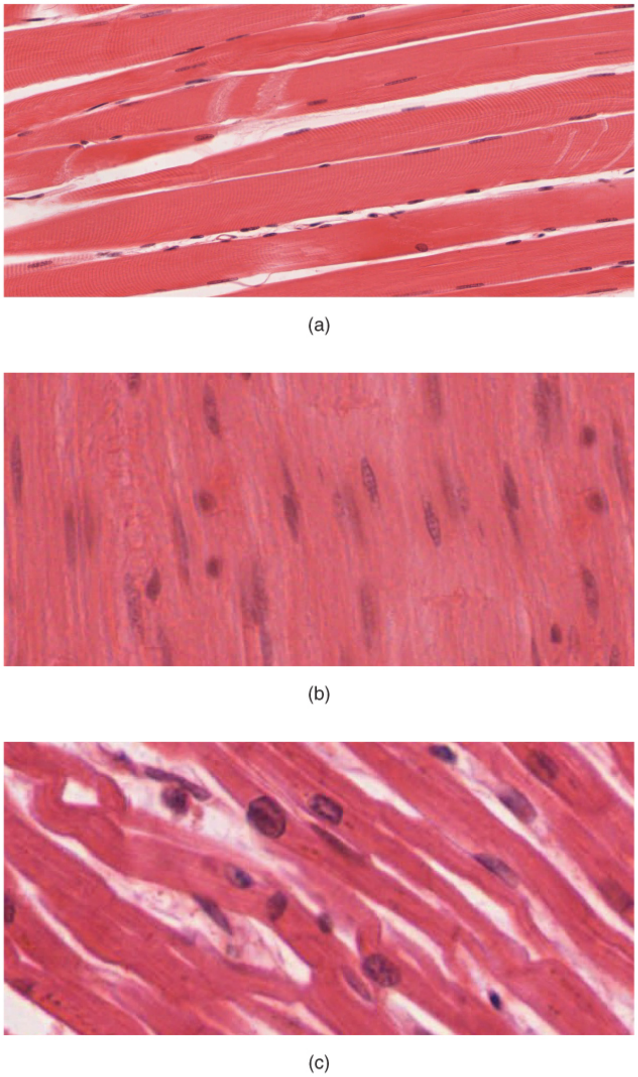
Muscular System Introduction

When most people think of muscles, they think of the muscles that are visible just under the skin, particularly of the limbs. These are skeletal muscles, so-named because most of them move the skeleton. But there are two other types of muscle in the body, with distinctly different jobs. Cardiac muscle, found in the heart, is concerned with pumping blood through the circulatory system. Smooth muscle is concerned with various involuntary movements, such as having one’s hair stand on end when cold or frightened, or moving food through the digestive system. This chapter will examine the structure and function of these three types of muscles.

The Three Types of Muscle

Muscle is one of the four primary tissue types of the body, and the body contains three types of muscle tissue: **skeletal muscle, cardiac muscle, and smooth muscle** ([link]). All three muscle tissues have some properties in common; they all exhibit a quality called **excitability** as their plasma membranes can change their electrical states (from polarized to depolarized) and send an electrical wave called an action potential along the entire length of the membrane. It is this change that signals the muscle cell to contract (shorten).

The Three Types of Muscle Tissue



The body contains three types of muscle tissue: (a) skeletal muscle, (b) smooth muscle, and (c) cardiac muscle. From top, LM × 1600, LM × 1600, LM × 1600. (Micrographs provided by the Regents of University of Michigan Medical School © 2012)

Muscles begin the actual process of contracting (shortening) when proteins in the muscle pull towards each other resulting in overlap and shortening. It is the action potential stimulated by the nervous system that allow this shortening to occur. All three muscle types have this property known as **contractility** which allows the cells to shorten with force.

When the signal from the nervous system ceases, a muscle can return to its original length due to a quality of muscle tissue called **elasticity**. It can recoil back to its original length due to elastic fibers. Muscle tissue also has the quality of **extensibility**; it can stretch or extend without tearing.

Differences among the three muscle types include the microscopic organization of their contractile proteins—**actin and myosin**. The actin and myosin proteins are arranged very regularly in the cytoplasm of individual muscle cells (referred to as fibers) in both skeletal muscle and cardiac muscle, which creates a pattern, or stripes, called **striations**. The striations are visible with a light microscope under high magnification (see [link]). **Skeletal muscle** cells (also called fibers) have many nuclei (sing. nucleus)so are called **multinucleated**. **Cardiac muscle** fibers each have one to two nuclei and are physically and electrically connected to each other so that the entire heart contracts as one unit (called a syncytium).

Because the actin and myosin are not arranged in such regular fashion in **smooth muscle**, the cytoplasm of a smooth muscle fiber (which has only a single nucleus) has a uniform, nonstriated appearance (resulting in the name smooth muscle). Smooth muscle in the walls of arteries is a critical component that regulates blood pressure necessary to push blood through the circulatory system; and smooth muscle in the skin, visceral organs, and internal passageways is essential for moving all materials through the body.

Glossary

cardiac muscle	striated muscle found in the heart; joined to one another at intercalated discs and under the regulation of pacemaker cells, which contract as one unit to pump blood through the circulatory system. Cardiac muscle is under involuntary control.
contractility	

ability to shorten (contract) forcibly

elasticity
ability to stretch and rebound

excitability
ability to undergo neural stimulation

extensibility
ability to lengthen (extend)

skeletal muscle
striated, multinucleated muscle that requires signaling from the nervous system to trigger contraction; most skeletal muscles are referred to as voluntary muscles that move bones and produce movement

smooth muscle
nonstriated, mononucleated muscle in the skin that is associated with hair follicles; assists in moving materials in the walls of internal organs, blood vessels, and internal passageways

Muscular System Module 2: Skeletal Muscle
By the end of this section, you will be able to:

- Describe the layers of connective tissues packaging skeletal muscle
- Explain how muscles work with tendons to move the body
- Identify areas of the skeletal muscle fibers
- Describe excitation-contraction coupling

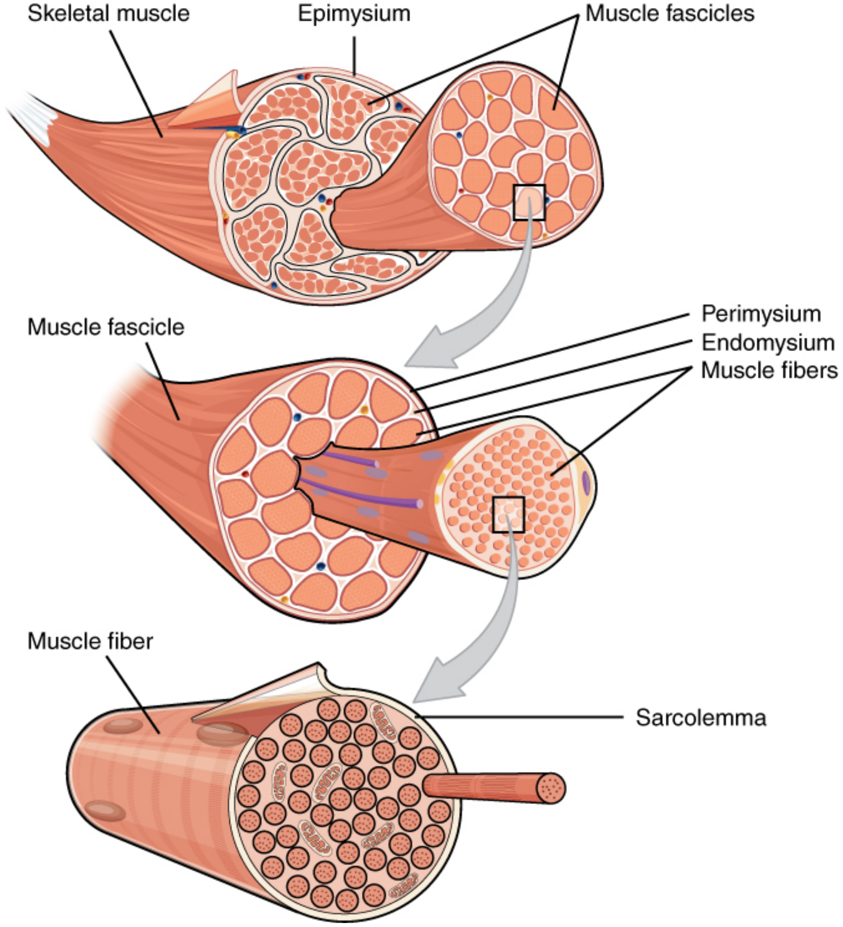
Skeletal Muscle

The best-known feature of skeletal muscle is its ability to contract and cause movement. Skeletal muscles act not only to produce movement but also to stop movement, such as resisting gravity to maintain posture. Small, constant adjustments of the skeletal muscles are needed to hold a body upright or balanced in any position. Muscles also prevent excess movement of the bones and joints, maintaining skeletal stability and preventing skeletal structure damage or deformation. Joints can become misaligned or dislocated entirely by pulling on the associated bones; muscles work to keep joints stable. Skeletal muscles are located throughout the body at the openings of internal tracts to control the movement of various substances. These muscles allow functions, such as swallowing, urination, and defecation, to be under voluntary control. Skeletal muscles also protect internal organs (particularly abdominal and pelvic organs) by acting as an external barrier or shield to external trauma and by supporting the weight of the organs.

Skeletal muscles contribute to the maintenance of **homeostasis** in the body by generating heat. Muscle contraction requires energy, and our bodies use ATP for energy. **ATP** When ATP is broken down, heat is produced. This heat is very noticeable during exercise, when sustained muscle movement causes body temperature to rise, and in cases of extreme cold, when shivering produces random skeletal muscle contractions to generate heat.

Each skeletal muscle is an organ that consists of various integrated tissues. These tissues include the skeletal muscle fibers, blood vessels, nerve fibers, and connective tissue. Each skeletal muscle has three layers of connective tissue that enclose it and provide structure to the muscle as a whole, and also compartmentalize the muscle fibers within the muscle ([link]). Each muscle is wrapped in a sheath of dense, irregular connective tissue called the **epimysium**, which allows a muscle to contract and move powerfully while maintaining its structural integrity. The epimysium also separates muscle from other tissues and organs in the area, allowing the muscle to move independently.

The Three Connective Tissue Layers



Bundles of muscle fibers, called fascicles, are covered by the perimysium. Muscle fibers are covered by the endomysium.

Inside each skeletal muscle, muscle fibers are organized into individual bundles, each called a **fascicle**, Each fascicle in surrounded by a middle layer of connective tissue called the **perimysium**. Inside each fascicle, each muscle fiber is encased in a thin connective tissue layer of connective tissue called the **endomysium**. The endomysium contains the nutrients to support the muscle fiber. These nutrients are supplied via blood to the muscle tissue.

In skeletal muscles that work with tendons to pull on bones, the collagen in the three tissue layers intertwines with the collagen of a tendon. At the other end of the tendon, it fuses with the periosteum coating the bone. The tension created by contraction of the muscle fibers is then transferred though the to the tendon, and then to the periosteum to pull on the bone for movement of the skeleton. In other places, the connective tissue may fuse with a broad, tendon-like sheet called an **aponeurosis**, or to fascia, the connective tissue between skin and bones. The broad sheet of connective tissue in the lower back that the latissimus dorsi muscles (the "lats") fuse into is an example of an aponeurosis.

Every skeletal muscle is also richly supplied by blood vessels for nourishment, oxygen delivery, and waste removal. In addition, every muscle fiber in a skeletal muscle is supplied by the axon branch of a **motor neuron**, which signals the fiber to contract. Unlike cardiac and smooth muscle, the only way to functionally contract a skeletal muscle is through signaling from the nervous system.

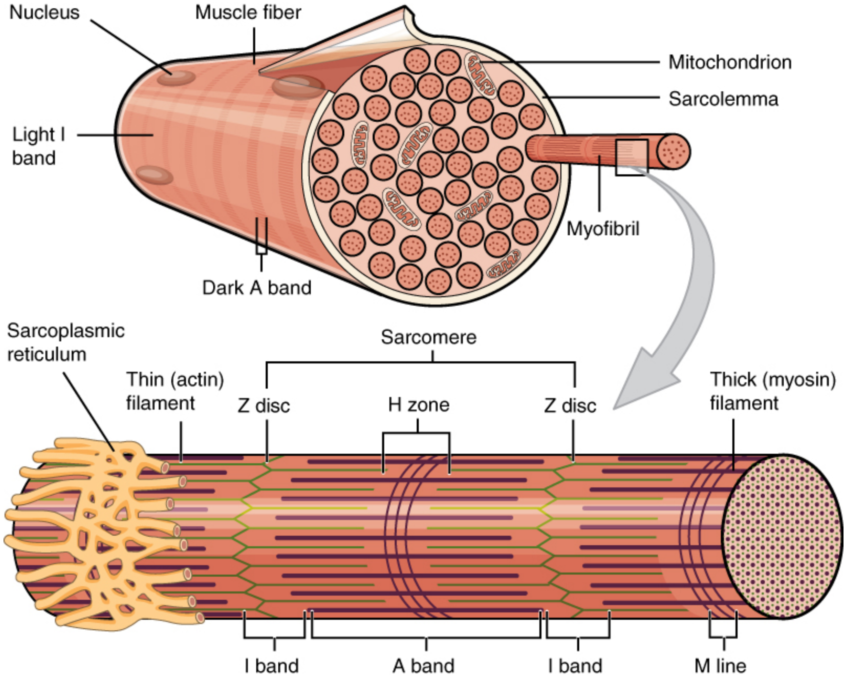
Skeletal Muscle Fibers

Because skeletal muscle cells are long and cylindrical, they are commonly referred to as muscle **fibers**. Skeletal muscle fibers can be quite large for human cells, with diameters up to 100 μm and lengths up to 30 cm (11.8 in) in the Sartorius of the upper leg. During early development, **myoblasts**, each with its own nucleus, fuse with up to hundreds of other myoblasts to form the **multinucleated** skeletal muscle fibers. Multiple nuclei permit the production of the large amounts of proteins and enzymes needed for muscle contraction.

Some other terminology associated with muscle fibers is rooted in the Greek *sarco*, which means "flesh." The plasma membrane of muscle fibers is called the **sarcolemma**, the cytoplasm is referred to as **sarcoplasm**, and the specialized smooth endoplasmic reticulum, is called the **sarcoplasmic reticulum (SR)** ([link]). As will soon be described, the **functional unit of a**

skeletal muscle fiber is the sarcomere, a highly organized arrangement of the contractile myofilaments **actin** (thin filament) and **myosin** (thick filament), along with other support proteins.

Muscle Fiber



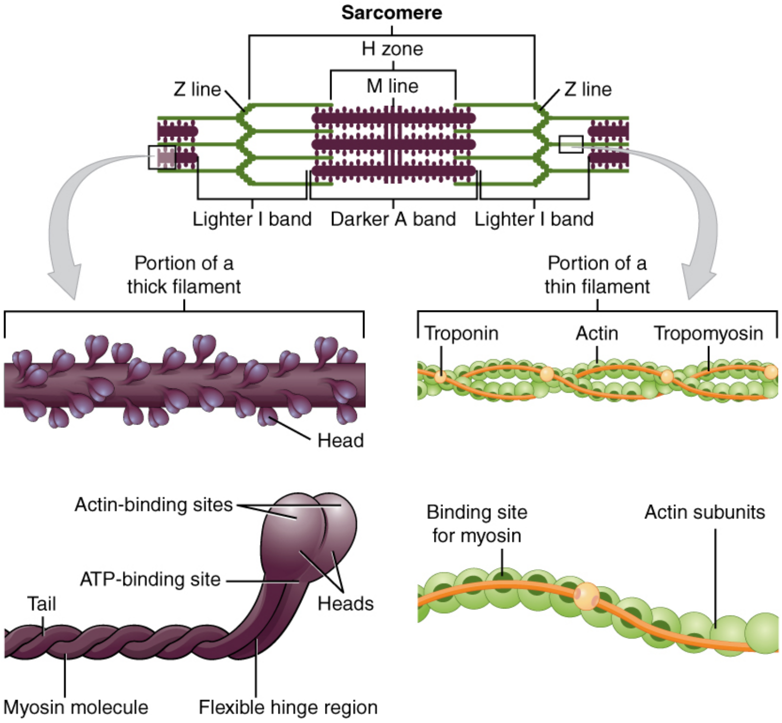
A skeletal muscle fiber is surrounded by a plasma membrane called the sarcolemma, which contains sarcoplasm, the cytoplasm of muscle cells. A muscle fiber is composed of many fibrils, which give the cell its striated appearance.

The Sarcomere

The striated appearance of skeletal muscle fibers is due to the arrangement of the **myofilaments of actin and myosin** in order from one end of the muscle fiber to the other. Actin is often referred to as the thin filament, while myosin may be called the thick filament.

The sarcomere is the **functional unit of the muscle fiber**. The sarcomere itself is bundled within the myofibril that runs the entire length of the muscle fiber. As myofibrils contract, the entire muscle cell contracts. Hundreds to thousands (each with thousands of sarcomeres) can be found inside one muscle fiber. Each sarcomere is bordered by structures called **Z-discs** (also called Z-lines), to which the actin myofilaments are anchored ([link]).

The Sarcomere



The sarcomere, the region from one Z-line to the next Z-line, is the functional unit of a skeletal muscle fiber.

The Neuromuscular Junction ***************************Module 2B starts here***********************************

Another specialization of the skeletal muscle is the site where a motor neuron’s terminal meets the muscle fiber—called the **neuromuscular**

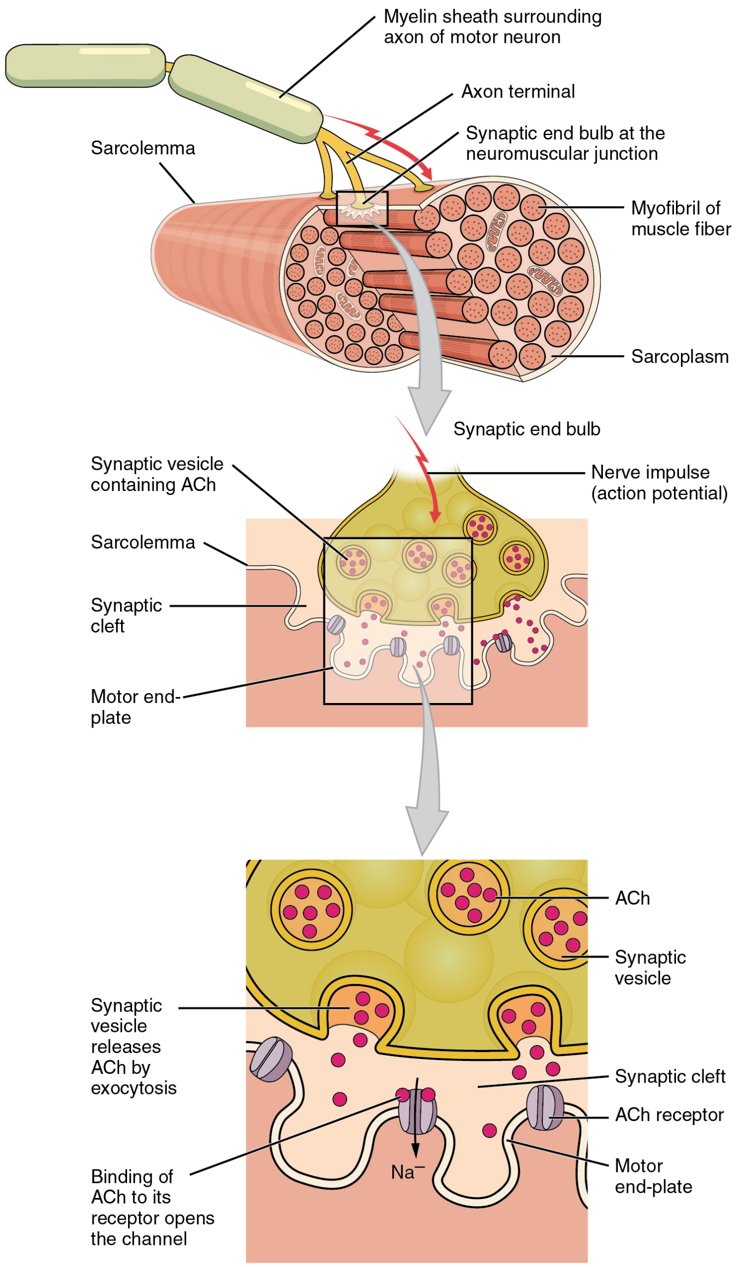
junction (NMJ). This is where the muscle fiber first responds to signaling by the motor neuron. Every skeletal muscle fiber in every skeletal muscle is innervated by a motor neuron at the NMJ. Excitation signals from the neuron are the only way to functionally activate the fiber to contract.

Stimulation of the Skeletal Muscle

All living cells have membrane potentials. Neurons and muscle cells can use their membrane potentials to generate electrical signals. They do this by controlling the movement of charged particles, called ions, across their membranes to create electrical currents. This is achieved by opening and closing specialized proteins in the membrane called ion channels. Although the currents generated by ions moving through these channel proteins are very small, they form the basis of both neural signaling and muscle contraction.

In skeletal muscle, this sequence begins with signals from the somatic motor division of the nervous system. In other words, the “excitation” step in skeletal muscles is always triggered by signaling from the nervous system ([link]).

Motor End-Plate and Innervation


Myelin sheath surrounding axon of motor neuron
Axon terminal
Synaptic end bulb at the neuromuscular junction
Sarcolemma
Myofibril of muscle fiber
Sarcoplasm
Synaptic end bulb
Nerve impulse (action potential)
Synaptic vesicle containing ACh
Sarcolemma
Synaptic cleft
Motor end-plate
ACh
Synaptic vesicle
Synaptic vesicle releases ACh by exocytosis
Synaptic cleft
ACh receptor
Na^{-}
Motor end-plate
Binding of ACh to its receptor opens the channel

At the NMJ, the axon terminal releases ACh. The motor end-plate is the location of the ACh-receptors in the muscle fiber sarcolemma. When ACh molecules are released, they diffuse across a minute space called the synaptic cleft and bind to the receptors.

The motor neurons that tell the skeletal muscle fibers to contract originate in the spinal cord, with a smaller number located in the brainstem for activation of skeletal muscles of the face, head, and neck. These neurons have long processes, called axons, which are specialized to transmit action potentials long distances— in this case, all the way from the spinal cord to the muscle itself (which may be up to three feet away). The axons of multiple neurons bundle together to form nerves, like wires bundled together in a cable.

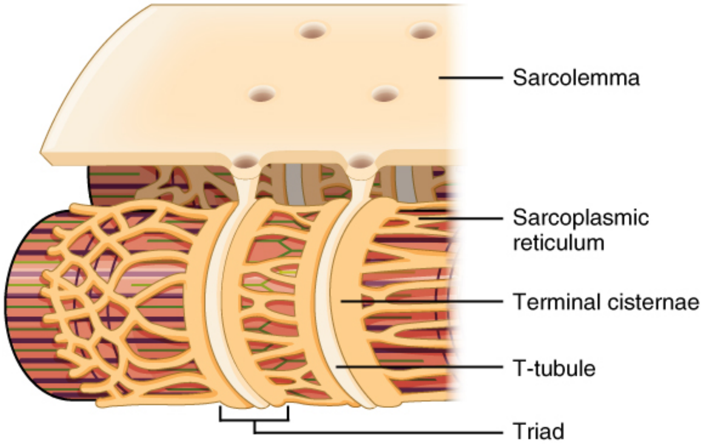
Signaling begins when a neuronal **action potential** travels along the axon of a motor neuron, and then along the individual branches to terminate at the NMJ. At the NMJ, the axon terminal releases a chemical messenger, or **neurotransmitter**, called **acetylcholine (ACh)**. The ACh molecules diffuse across a minute space called the **synaptic cleft** and bind to ACh receptors located within the **motor end-plate** of the sarcolemma on the other side of the synapse. Once ACh binds, a channel in the ACh receptor opens and positively charged ions can pass through into the muscle fiber, causing it to **depolarize**, meaning that the membrane potential of the muscle fiber becomes less negative (closer to zero.)

As the membrane depolarizes, another set of ion channels are triggered to open. Ions enter the muscle fiber, and an action potential rapidly spreads (or “fires”) along the entire membrane to begin the process of muscle contraction.

Things happen very quickly in the world of excitable membranes (just think about how quickly you can snap your fingers as soon as you decide to do it). Immediately following depolarization of the membrane, it repolarizes, re-establishing the normal state. Meanwhile, the ACh in the synaptic cleft is degraded by the enzyme **acetylcholinesterase (AChE)** so that the ACh cannot rebind to a receptor and cause unwanted extended muscle excitation and contraction.

The signal is sent along the sarcolemma which triggers the release of calcium ions (Ca^{++}) from its storage in the cell’s sarcoplasmic reticulum. **T-tubules** bring that signal to the SR. The arrangement of a T-tubule with the membranes of SR on either side is called a **triad** ([link]). The triad surrounds the cylindrical structure called a **myofibril**, which contains actin and myosin.

The T-tubule



Narrow T-tubules permit the conduction of electrical impulses. The SR functions to regulate intracellular levels of calcium. Two terminal cisternae (where enlarged SR connects to the T-tubule) and one T-tubule comprise a triad—a “threesome” of membranes, with those of SR on two sides and the T-tubule sandwiched between them.

The T-tubules carry the action potential into the interior of the cell, which triggers the opening of calcium channels in the membrane of the adjacent SR, causing Ca^{++} to diffuse out of the SR and into the sarcoplasm. It is the arrival of Ca^{++} in the sarcoplasm that initiates contraction of the muscle fiber by its contractile units, or sarcomeres.

Chapter Review

Skeletal muscles contain connective tissue, blood vessels, and nerves. There are three layers of connective tissue: epimysium, perimysium, and endomysium. Skeletal muscle fibers are organized into groups called fascicles. Blood vessels and nerves enter the connective tissue and branch in the cell. Muscles attach to bones directly or through tendons or aponeuroses. Skeletal muscles maintain posture, stabilize bones and joints, control internal movement, and generate heat.

Skeletal muscle fibers are long, multinucleated cells. The membrane of the cell is the sarcolemma; the cytoplasm of the cell is the sarcoplasm. The sarcoplasmic reticulum (SR) is a form of endoplasmic reticulum. Muscle fibers are composed of myofibrils. The striations are created by the organization of actin and myosin resulting in the banding pattern of myofibrils.

Glossary

acetylcholine (ACh)

neurotransmitter that binds at a motor end-plate to trigger depolarization

actin

protein that makes up most of the thin myofilaments in a sarcomere muscle fiber

action potential

change in voltage of a cell membrane in response to a stimulus that results in transmission of an electrical signal; unique to neurons and muscle fibers

aponeurosis

broad, tendon-like sheet of connective tissue that attaches a skeletal muscle to another skeletal muscle or to a bone

depolarize

to reduce the voltage difference between the inside and outside of a cell’s plasma membrane (the sarcolemma for a muscle fiber), making the inside less negative than at rest

endomysium
loose, and well-hydrated connective tissue covering each muscle fiber in a skeletal muscle

epimysium
outer layer of connective tissue around a skeletal muscle

excitation-contraction coupling
sequence of events from motor neuron signaling to a skeletal muscle fiber to contraction of the fiber’s sarcomeres

fascicle
bundle of muscle fibers within a skeletal muscle

motor end-plate
sarcolemma of muscle fiber at the neuromuscular junction, with receptors for the neurotransmitter acetylcholine

myofibril
long, cylindrical organelle that runs parallel within the muscle fiber and contains the sarcomeres

myosin
protein that makes up most of the thick cylindrical myofilament within a sarcomere muscle fiber

neuromuscular junction (NMJ)
synapse between the axon terminal of a motor neuron and the section of the membrane of a muscle fiber with receptors for the acetylcholine released by the terminal

neurotransmitter
signaling chemical released by nerve terminals that bind to and activate receptors on target cells

perimysium

connective tissue that bundles skeletal muscle fibers into fascicles within a skeletal muscle

sarcomere
longitudinally, repeating functional unit of skeletal muscle, with all of the contractile and associated proteins involved in contraction

sarcolemma
plasma membrane of a skeletal muscle fiber

sarcoplasm
cytoplasm of a muscle cell

sarcoplasmic reticulum (SR)
specialized smooth endoplasmic reticulum, which stores, releases, and retrieves Ca^{++}

synaptic cleft
space between a nerve (axon) terminal and a motor end-plate

T-tubule
projection of the sarcolemma into the interior of the cell

thick filament
the thick myosin strands and their multiple heads projecting from the center of the sarcomere toward, but not all to way to, the Z-discs

thin filament
thin strands of actin and its troponin-tropomyosin complex projecting from the Z-discs toward the center of the sarcomere

triad
the grouping of one T-tubule and two terminal cisternae

troponin
regulatory protein that binds to actin, tropomyosin, and calcium

tropomyosin

regulatory protein that covers myosin-binding sites to prevent actin from binding to myosin

voltage-gated sodium channels

membrane proteins that open sodium channels in response to a sufficient voltage change, and initiate and transmit the action potential as Na^+ enters through the channel

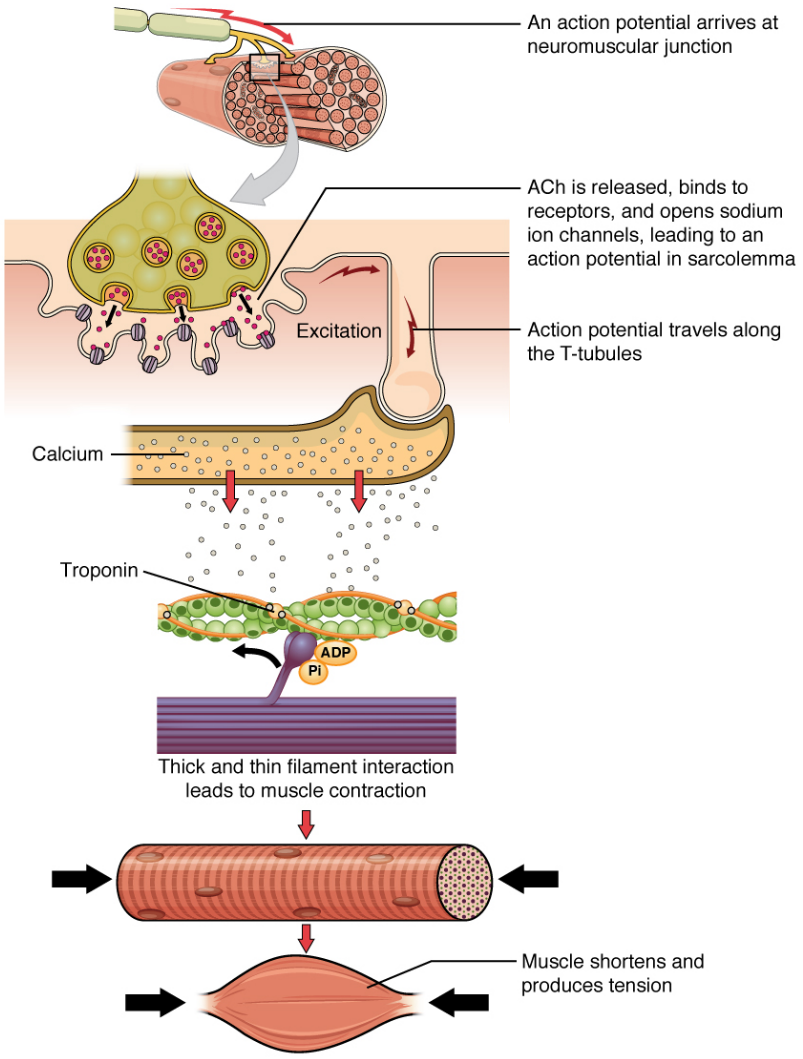
Muscular System Module 3: Contraction and Relaxation

By the end of this section, you will be able to:

- Describe the components involved in a muscle contraction
- Explain how muscles contract and relax
- Describe the sliding filament model of muscle contraction

The sequence of events that result in the contraction of an individual muscle fiber begins with a signal, the neurotransmitter, ACh—from the motor neuron innervating that fiber. The local membrane of the fiber will depolarize, triggering an action potential that spreads to the rest of the membrane, including the T-tubules. This triggers the release of calcium ions (Ca^{++}) from storage in the sarcoplasmic reticulum (SR). The Ca^{++} then initiates contraction, which is sustained with energy supplied by **ATP** ([link]). A molecule named **troponin** covers the active sites on the myosin. The active site is where actin binds to myosin as part of the contraction process. As long as Ca^{++} ions remain in the sarcoplasm to bind to troponin, which keeps the actin-binding sites “unshielded,” and as long as ATP is available to drive the cross-bridge cycling and the pulling of actin strands by myosin, the muscle fiber will continue to shorten to an anatomical limit.

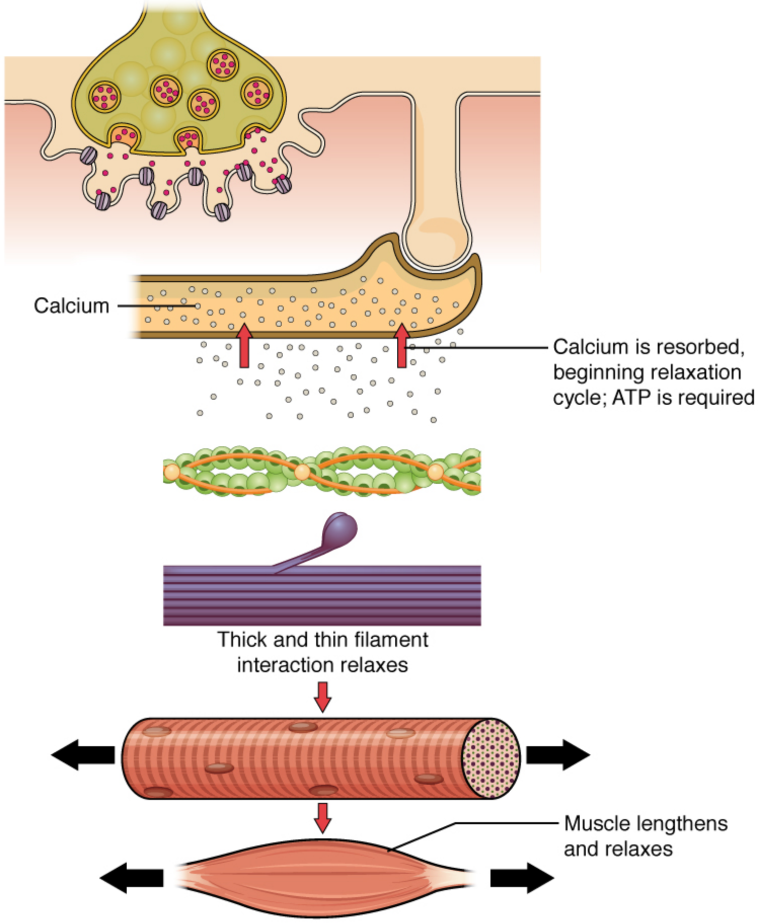
Contraction of a Muscle Fiber


An action potential arrives at neuromuscular junction
ACh is released, binds to receptors, and opens sodium ion channels, leading to an action potential in sarcolemma
Excitation
Action potential travels along the T-tubules
Calcium
Troponin
ADP
Pi
Thick and thin filament interaction leads to muscle contraction
Muscle shortens and produces tension

A cross-bridge forms between actin and the myosin heads triggering contraction. As long as Ca^{++} ions remain in the sarcoplasm to bind to troponin, and as long as ATP is available, the muscle fiber will continue to shorten.

Muscle contraction usually stops when signaling from the motor neuron ends, which repolarizes the sarcolemma and T-tubules. Ca^{++} ions are then pumped back into the SR, which causes the troponin to reshield (or recover) the binding sites on the actin strands. A muscle also can stop contracting when it runs out of ATP and becomes fatigued ([link]).

Relaxation of a Muscle Fiber


Calcium
Calcium is resorbed, beginning relaxation cycle; ATP is required
Thick and thin filament interaction relaxes
Muscle lengthens and relaxes

Ca^{++} ions are pumped back into the SR, which causes the tropomyosin to reshield the binding sites on the actin strands. A muscle may also stop contracting when it runs out of ATP and becomes fatigued.

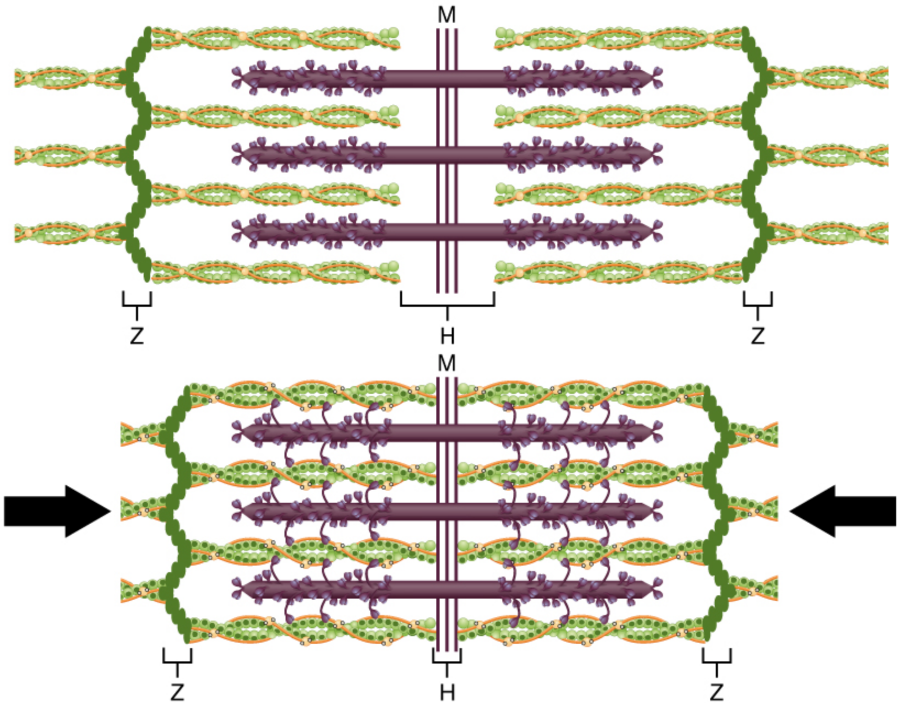
The molecular events of muscle fiber shortening occur within the fiber’s sarcomeres (see [link]). The contraction of a striated muscle fiber occurs as the sarcomeres, arranged within myofibrils, shorten as myosin heads pull on the actin filaments.

The region where thick and thin filaments overlap has a dense appearance, as there is little space between the filaments. This zone where thin and thick filaments overlap is very important to muscle contraction, as it is the site where filament movement starts. Thin filaments, anchored at their ends by the Z-discs, do not extend completely into the central region that only contains thick filaments, anchored at their bases at a spot called the M-line. A myofibril is composed of many sarcomeres running along its length; thus, myofibrils and muscle cells contract as the sarcomeres contract.

The Sliding Filament Model of Contraction

When signaled by a motor neuron, a skeletal muscle fiber contracts as the thin filaments are pulled and then slide past the thick filaments within the fiber's sarcomeres. This causes the z lines to be pulled closer together. It is important to note that the proteins actin and myosin don't lengthen or shorten, but they increase the area of overlap. If you think about a telescoping umbrella handle you can envision the handle becoming shorter as you close the umbrella, with the parts sliding over each other even though none of the handle's parts get shorter. This process is known as the **sliding filament model of muscle contraction** ([link]). The sliding can only occur when myosin-binding sites on the actin filaments are exposed by a series of steps that begins with Ca^{++} entry into the sarcoplasm.

The Sliding Filament Model of Muscle Contraction



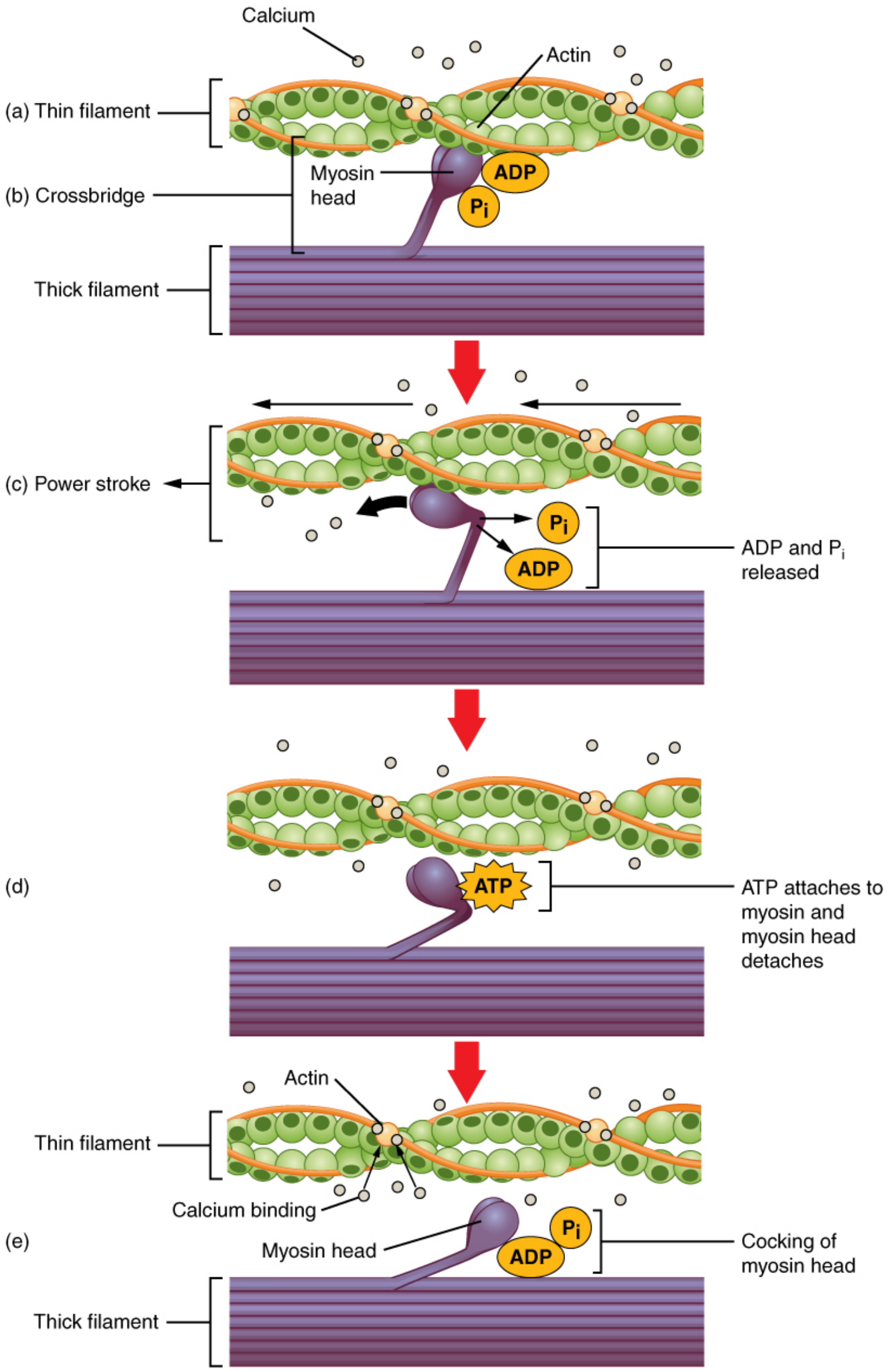
When a sarcomere contracts, the Z lines move closer together, and the I band becomes smaller. The A band stays the same width. At full contraction, the thin and thick filaments overlap.

Tropomyosin is a protein that winds around the chains of the actin filament and covers the myosin-binding sites to prevent actin from binding to myosin. Tropomyosin binds to troponin to form a troponin-tropomyosin complex. The **troponin-tropomyosin complex** prevents the myosin “heads” from binding to the active sites on the actin microfilaments. Troponin also has a binding site for Ca^{++} ions.

To initiate muscle contraction, tropomyosin has to expose the **myosin binding sites**. This allows the myosin heads to bind to these exposed binding sites and form **cross-bridges**. The thin filaments are then pulled by the myosin heads to slide past the thick filaments toward the center of the sarcomere. But each head can only pull a very short distance before it has reached its limit and must be “re-cock” before it can pull again, a step that requires ATP.

ATP and Muscle Contraction

For thin filaments to continue to slide past thick filaments during muscle contraction, myosin heads must pull the actin at the binding sites, detach, re-cock, attach to more binding sites, pull, detach, re-cock, etc. This repeated movement is known as the cross-bridge cycle. This motion of the myosin heads is similar to the oars when an individual rows a boat: The paddle of the oars (the myosin heads) pull, are lifted from the water (detach), repositioned (re-cocked) and then immersed again to pull ([link]). Each cycle requires energy, and the action of the myosin heads in the sarcomeres repetitively pulling on the thin filaments also requires energy, which is provided by ATP.
Skeletal Muscle Contraction



(a) The active site on actin is exposed as calcium binds to troponin. (b) The myosin head is attracted to actin, and myosin binds actin at its actin-binding site, forming the cross-bridge. (c) During the power stroke, the phosphate generated in the previous contraction cycle is released. This results in the myosin head pivoting toward the center of the sarcomere, after which the attached ADP and phosphate group are released. (d) A new molecule of ATP attaches to the myosin head, causing the cross-bridge to detach. (e) The myosin head hydrolyzes ATP to ADP and phosphate, which returns the myosin to the cocked position.

Cross-bridge formation occurs when the myosin head attaches to the actin while adenosine diphosphate (ADP) and inorganic phosphate (P_i) are still bound to myosin ([link]**a,b**). P_i is then released, causing myosin to form a stronger attachment to the actin, after which the myosin head moves toward the M-line, pulling the actin along with it. As actin is pulled, the filaments move approximately 10 nm toward the M-line. This movement is called the **power stroke**, as movement of the thin filament occurs at this step ([link]**c**). In the absence of ATP, the myosin head will not detach from actin.

One part of the myosin head attaches to the binding site on the actin, but the head has another binding site for ATP. ATP binding causes the myosin head to detach from the actin ([link]**d**). After this occurs, ATP is converted to ADP and P_i by the intrinsic **ATPase** activity of myosin. The energy released during ATP hydrolysis changes the angle of the myosin head into a cocked position ([link]**e**). The myosin head is now in position for further movement.

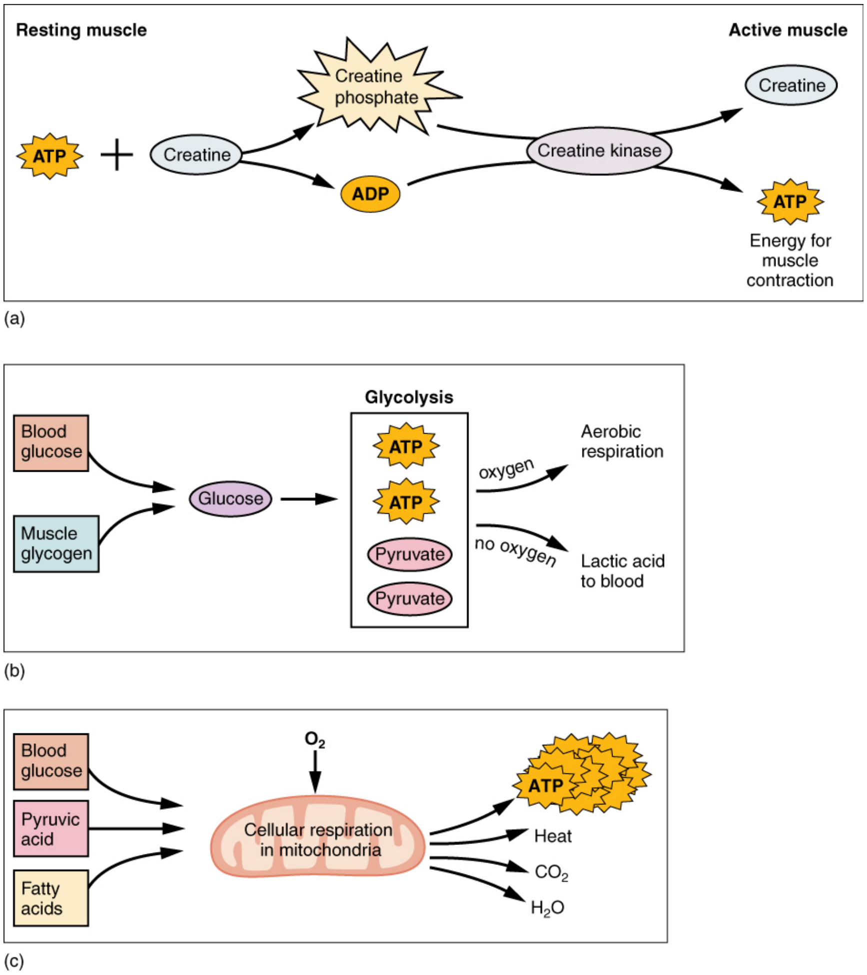
When the myosin head is cocked, myosin is in a high-energy configuration. This energy is expended as the myosin head moves through the power stroke, and at the end of the power stroke, the myosin head is in a low-energy position. After the power stroke, ADP is released; however, the formed cross-bridge is still in place, and actin and myosin are bound together. As long as ATP is available, it readily attaches to myosin, the cross-bridge cycle can recur, and muscle contraction can continue.

Note that each thick filament of roughly 300 myosin molecules has multiple myosin heads, and many cross-bridges form and break continuously during muscle contraction. Multiply this by all of the sarcomeres in one myofibril, all the myofibrils in one muscle fiber, and all of the muscle fibers in one skeletal muscle, and you can understand why so much energy (ATP) is needed to keep skeletal muscles working. In fact, it is the loss of ATP that results in the rigor mortis observed soon after someone dies. With no further ATP production possible, there is no ATP available for myosin heads to detach from the actin-binding sites, so the cross-bridges stay in place, causing the rigidity in the skeletal muscles.

Sources of ATP

ATP supplies the energy for muscle contraction to take place. In addition to its direct role in the cross-bridge cycle, ATP also provides the energy for the active-transport Ca^{++} pumps in the SR. Muscle contraction does not occur without sufficient amounts of ATP. The amount of ATP stored in muscle is very low, only sufficient to power a few seconds worth of contractions. As it is broken down, ATP must therefore be regenerated and replaced quickly to allow for sustained contraction. There are three mechanisms by which ATP can be regenerated: **creatine phosphate metabolism, anaerobic glycolysis, fermentation and aerobic respiration** .

Creatine phosphate is a molecule that can store energy in its phosphate bonds. In a resting muscle, excess ATP transfers its energy to creatine, producing ADP and creatine phosphate. This acts as an energy reserve that can be used to quickly create more ATP. When the muscle starts to contract and needs energy, creatine phosphate transfers its phosphate back to ADP to form ATP and creatine. ([link]).
Muscle Metabolism



(a) Some ATP is stored in a resting muscle. As contraction starts, it is used up in seconds. More ATP is generated from creatine phosphate for about 15 seconds. (b) Each glucose molecule produces two ATP and two molecules of pyruvic acid, which can be used in aerobic respiration or converted to lactic acid. If oxygen is not available, pyruvic acid is converted to lactic acid, which may contribute to muscle fatigue. This occurs during strenuous exercise when high amounts of energy are needed but oxygen cannot be sufficiently delivered to muscle. (c) Aerobic respiration is the breakdown of glucose in the presence of oxygen (O_2) to produce carbon dioxide, water, and ATP.

Approximately 95 percent of the ATP required for resting or moderately active muscles is provided by aerobic respiration, which takes place in mitochondria.

Muscle fatigue occurs when a muscle can no longer contract in response to signals from the nervous system. The exact causes of muscle fatigue are not fully known, although certain factors have been correlated with the decreased muscle contraction that occurs during fatigue. ATP is needed for normal muscle contraction, and as ATP reserves are reduced, muscle function may decline.

Relaxation of a Skeletal Muscle

Relaxing skeletal muscle fibers, and ultimately, the skeletal muscle, begins with the motor neuron, which stops releasing its chemical signal, ACh, into the synapse at the NMJ. The muscle fiber will repolarize, which closes the gates in the SR where Ca^{++} was being released. ATP-driven pumps will move Ca^{++} out of the sarcoplasm back into the SR. This results in the “reshielding” of the actin-binding sites on the thin filaments. Without the ability to form cross-bridges between the thin and thick filaments, the muscle fiber loses its tension and relaxes.

Muscle Strength

The number of skeletal muscle fibers in a given muscle is genetically determined and **does not change** . Muscle strength is directly related to the amount of myofibrils and sarcomeres within each fiber. Factors, such as hormones and stress (and artificial anabolic steroids), acting on the muscle can increase the production of sarcomeres and myofibrils within the muscle fibers, a change called **hypertrophy** , which results in the increased mass and bulk in a skeletal muscle. Likewise, decreased use of a skeletal muscle results in **atrophy** , where the number of sarcomeres and myofibrils disappear (but not the number of muscle fibers). It is common for a limb in a cast to show atrophied muscles when the cast is removed, and certain diseases, such as polio, show atrophied muscles.

Note:

Disorders of the …

Muscular System

Duchenne muscular dystrophy (DMD) is a progressive weakening of the skeletal muscles. It is one of several diseases collectively referred to as "muscular dystrophy." DMD is caused by a lack of the protein dystrophin, which helps the thin filaments of myofibrils bind to the sarcolemma. Over time, as muscle damage accumulates, muscle mass is lost, and greater functional impairments develop.

DMD is an inherited disorder caused by an **abnormal X chromosome** . It primarily affects males, and it is usually diagnosed in early childhood. DMD usually first appears as difficulty with balance and motion, and then progresses to an inability to walk. It continues progressing upward in the body from the lower extremities to the upper body, where it affects the muscles responsible for breathing and circulation. It ultimately causes death due to respiratory failure, and those afflicted do not usually live past their 20s.

Because DMD is caused by a mutation in the gene that codes for dystrophin, it was thought that introducing healthy myoblasts into patients might be an effective treatment. **Myoblasts** are the embryonic cells responsible for muscle development, and ideally, they would carry healthy genes that could produce the dystrophin needed for normal muscle contraction. This approach has been largely unsuccessful in humans. A recent approach has involved attempting to boost the muscle's production of utrophin, a protein similar to dystrophin that may be able to assume the role of dystrophin and prevent cellular damage from occurring.

Chapter Review

A sarcomere is the smallest contractile portion of a muscle. Myofibrils are composed of thick and thin filaments. Thick filaments are composed of the protein myosin; thin filaments are composed of the protein actin. Troponin and tropomyosin are regulatory proteins.

Muscle contraction is described by the sliding filament model of contraction. ACh is the neurotransmitter that binds at the neuromuscular junction (NMJ) to trigger depolarization, and an action potential travels along the sarcolemma to trigger calcium release from SR. The actin sites are exposed after Ca^{++} enters the sarcoplasm from its SR storage to activate the troponin-tropomyosin complex so that the tropomyosin shifts away from the sites. The cross-bridging of myposin heads docking into actin-binding sites is followed by the "power stroke"—the sliding of the thin filaments by thick filaments. The power strokes are powered by ATP. Ultimately, the sarcomeres, myofibrils, and muscle fibers shorten to produce movement.

Glossary

aerobic respiration
production of ATP in the presence of oxygen

ATPase
enzyme that hydrolyzes ATP to ADP

creatine phosphate
phosphagen used to store energy from ATP and transfer it to muscle

glycolysis
anaerobic breakdown of glucose to ATP

lactic acid
product of anaerobic glycolysis

oxygen debt
amount of oxygen needed to compensate for ATP produced without oxygen during muscle contraction

power stroke
action of myosin pulling actin inward (toward the M line)

pyruvic acid

product of glycolysis that can be used in aerobic respiration or converted to lactic acid

Muscular System Module 4: Nervous System Control of Muscle Tension

By the end of this section, you will be able to:

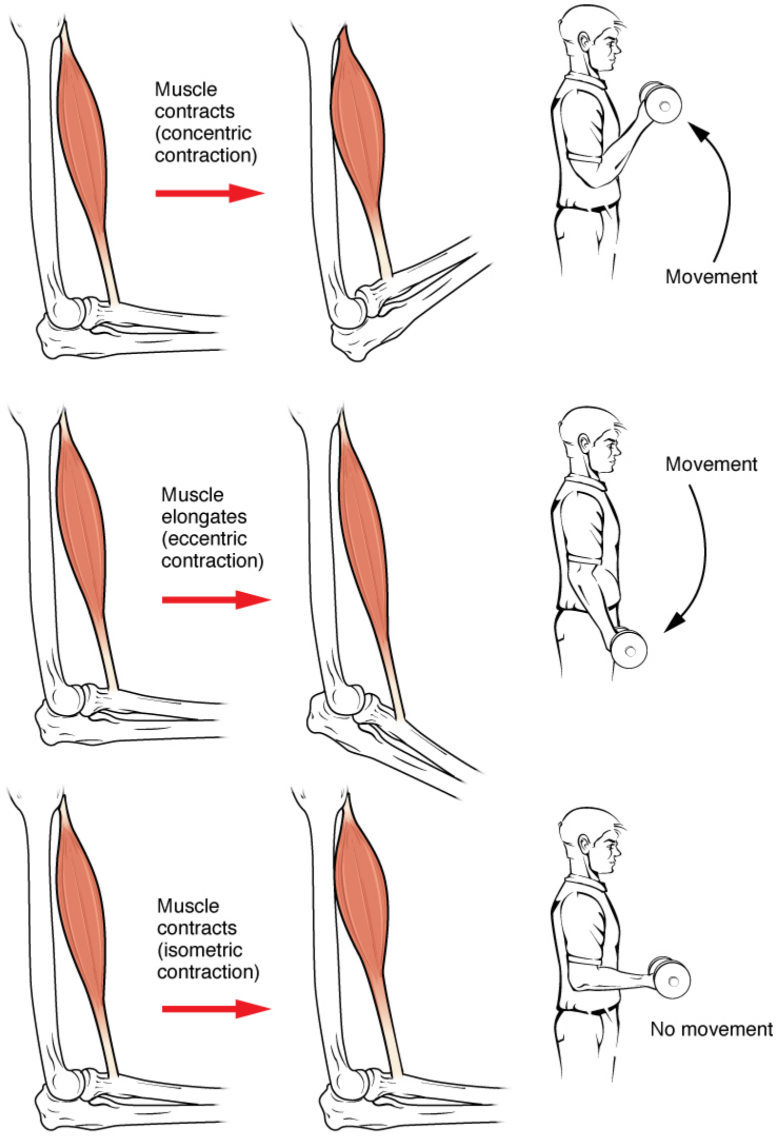
- Explain concentric, isotonic, and eccentric contractions
- Describe the length-tension relationship
- Describe the three phases of a muscle twitch
- Define wave summation, tetanus, and treppe

To move an object, referred to as load, the sarcomeres in the muscle fibers of the skeletal muscle must shorten. The force generated by the contraction of the muscle (or shortening of the sarcomeres) is called **muscle tension**. However, muscle tension also is generated when the muscle is contracting against a load that does not move, resulting in two main types of skeletal muscle contractions: isotonic contractions and isometric contractions.

In **isotonic contractions**, where the tension in the muscle stays constant, a load is moved as the length of the muscle changes (shortens). There are two types of isotonic contractions: concentric and eccentric. A **concentric contraction** involves the muscle shortening to move a load. An example of this is the biceps brachii muscle contracting when a hand weight is brought upward with increasing muscle tension. As the biceps brachii contract, the angle of the elbow joint decreases as the forearm is brought toward the body. Here, the biceps brachii contracts as sarcomeres in its muscle fibers are shortening and cross-bridges form; the myosin heads pull the actin. An **eccentric contraction** occurs as the muscle tension diminishes and the muscle lengthens. In this case, the hand weight is lowered in a slow and controlled manner as the amount of cross-bridges being activated by nervous system stimulation decreases. In this case, as tension is released from the biceps brachii, the angle of the elbow joint increases. Eccentric contractions are also used for movement and balance of the body.

An **isometric contraction** occurs as the muscle produces tension without changing the angle of a skeletal joint. Isometric contractions involve sarcomere shortening and increasing muscle tension, but do not move a load, as the force produced cannot overcome the resistance provided by the load. For example, if one attempts to lift a hand weight that is too heavy, there will be sarcomere activation and shortening to a point, and ever-increasing muscle tension, but no change in the angle of the elbow joint. In everyday living, isometric contractions are active in maintaining posture and maintaining bone and joint stability. However, holding your head in an upright position occurs not because the muscles cannot move the head, but because the goal is to remain stationary and not produce movement. Most actions of the body are the result of a combination of isotonic and isometric contractions working together to produce a wide range of outcomes ([link]).

Types of Muscle Contractions


Muscle contracts (concentric contraction)
Movement
Muscle elongates (eccentric contraction)
Movement
Muscle contracts (isometric contraction)
No movement

During isotonic contractions, muscle length changes to move a load. During isometric contractions, muscle length does not change because the load exceeds the tension the muscle can generate.

All of these muscle activities are under the exquisite control of the nervous system. Neural control regulates concentric, eccentric and isometric contractions, muscle fiber recruitment, and muscle tone. A crucial aspect of nervous system control of skeletal muscles is the role of motor units.

Motor Units

As you have learned, every skeletal muscle fiber must be innervated by the axon terminal of a motor neuron in order to contract. Each muscle fiber is innervated by only one motor neuron. The actual group of muscle fibers in a muscle innervated by a single motor neuron is called a **motor unit**. The size of a motor unit is variable depending on the nature of the muscle.

A **small motor unit** is an arrangement where a single motor neuron supplies a small number of muscle fibers in a muscle. Small motor units permit very fine motor control of the muscle. Small motor units are involved in the many fine movements of the fingers and thumb of the hand for grasping, texting, etc.

A **large motor unit** is an arrangement where a single motor neuron supplies a large number of muscle fibers in a muscle. Large motor units are concerned with simple, or “gross,” movements, such as powerfully extending the knee joint. The best example is the large motor units of the thigh muscles or back muscles, where a single motor neuron will supply thousands of muscle fibers in a muscle, as its axon splits into thousands of branches.

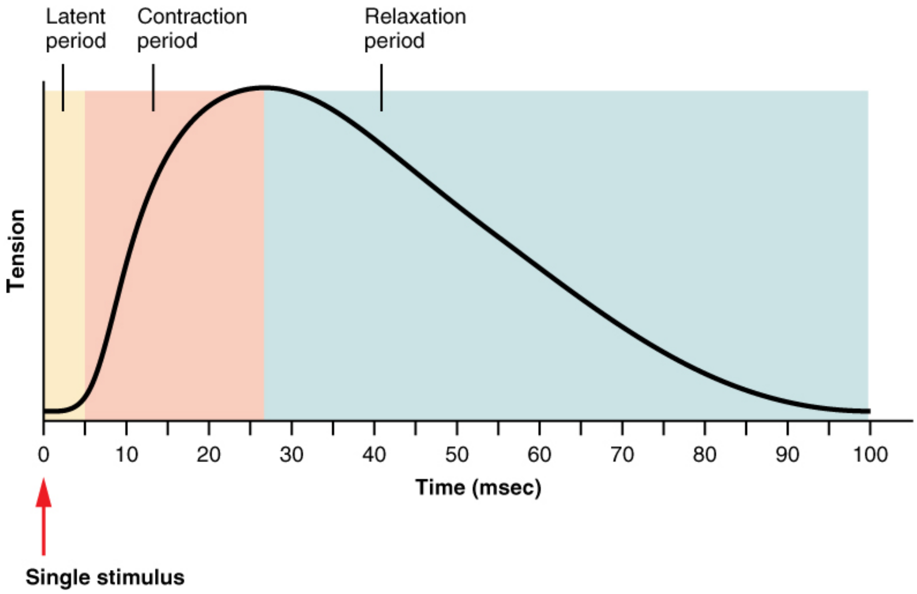
The increasing activation of motor units produces an increase in muscle contraction known as **recruitment**. As more motor units are recruited, the muscle contraction grows progressively stronger. In some muscles, the largest motor units may generate a contractile force of 50 times more than the smallest motor units in the muscle. This allows a feather to be picked up using the biceps brachii arm muscle with minimal force, and a heavy weight to be lifted by the same muscle by recruiting the largest motor units.

When necessary, the maximal number of motor units in a muscle can be recruited simultaneously, producing the maximum force of contraction for that muscle, but this cannot last for very long because of the energy requirements to sustain the contraction. To prevent complete muscle fatigue, motor units are generally not all simultaneously active, but instead some motor units rest while others are active, which allows for longer muscle contractions. The nervous system uses recruitment as a mechanism to efficiently utilize a skeletal muscle.

The Frequency of Motor Neuron Stimulation

A single action potential from a motor neuron will produce a single contraction in the muscle fibers of its motor unit. This isolated contraction is called a **twitch**. A twitch can last for a few milliseconds or 100 milliseconds, depending on the muscle type. The tension produced by a single twitch can be measured by a **myogram**, an instrument that measures the amount of tension produced over time ([link]). Each twitch undergoes three phases. The first phase is the **latent period**, during which the action potential is being propagated along the sarcolemma and Ca^{++} ions are released from the SR. This is the phase during which excitation and contraction are being coupled but contraction has yet to occur. The **contraction phase** occurs next. The Ca^{++} ions in the sarcoplasm have bound to troponin, tropomyosin has shifted away from actin-binding sites, cross-bridges formed, and sarcomeres are actively shortening to the point of peak tension. The last phase is the **relaxation phase**, when tension decreases as contraction stops. Ca^{++} ions are pumped out of the sarcoplasm into the SR, and cross-bridge cycling stops, returning the muscle fibers to their resting state.

A Myogram of a Muscle Twitch



A single muscle twitch has a latent period, a contraction phase when tension increases, and a relaxation phase when tension decreases. During the latent period, the action potential is being propagated along the sarcolemma. During the contraction phase, Ca^{++} ions in the sarcoplasm bind to troponin, tropomyosin moves from actin-binding sites, cross-bridges form, and sarcomeres shorten. During the relaxation phase, tension decreases as Ca^{++} ions are pumped out of the sarcoplasm and cross-bridge cycling stops.

Muscle Tone

Skeletal muscles are rarely completely relaxed, or flaccid. Even if a muscle is not producing movement, it is contracted a small amount to maintain its contractile proteins and produce **muscle tone**. The tension produced by muscle tone allows muscles to continually stabilize joints and maintain posture.

Muscle tone is accomplished by a complex interaction between the nervous system and skeletal muscles that results in the activation of a few motor units at a time, most likely in a cyclical manner. In this manner, muscles never fatigue completely, as some motor units can recover while others are active.

The absence of the low-level contractions that lead to muscle tone is referred to as **hypotonia** or atrophy, and can result from damage to parts of the central nervous system (CNS), such as the cerebellum, or from loss of innervations to a skeletal muscle, as in poliomyelitis. Hypotonic muscles have a flaccid appearance and display functional impairments, such as weak reflexes. Conversely, excessive muscle tone is referred to as **hypertonia**, accompanied by hyperreflexia (excessive reflex responses), often the result of damage to upper motor neurons in the CNS. Hypertonia can present with muscle rigidity (as seen in Parkinson's disease) or spasticity, a phasic change in muscle tone, where a limb will "snap" back from passive stretching (as seen in some strokes).

Chapter Review

The number of cross-bridges formed between actin and myosin determines the amount of tension produced by a muscle. The length of a sarcomere is optimal when the zone of overlap between thin and thick filaments is greatest. Muscles that are stretched or compressed too greatly do not produce maximal amounts of power. A motor unit is formed by a motor neuron and all of the muscle fibers that are innervated by that same motor neuron. A single contraction is called a twitch. A muscle twitch has a latent period, a contraction phase, and a relaxation phase. A graded muscle response allows variation in muscle tension. Summation occurs as successive stimuli are added together to produce a stronger muscle contraction. Tetanus is the fusion of contractions to produce a continuous contraction. Increasing the number of motor neurons involved increases the amount of motor units activated in a muscle, which is called recruitment. Muscle tone is the constant low-level contractions that allow for posture and stability.

Glossary

concentric contraction
muscle contraction that shortens the muscle to move a load

contraction phase
twitch contraction phase when tension increases

eccentric contraction
muscle contraction that lengthens the muscle as the tension is diminished

graded muscle response
modification of contraction strength

hypertonia
abnormally high muscle tone

hypotonia
abnormally low muscle tone caused by the absence of low-level contractions

isometric contraction
muscle contraction that occurs with no change in muscle length

isotonic contraction
muscle contraction that involves changes in muscle length

latent period
the time when a twitch does not produce contraction

motor unit
motor neuron and the group of muscle fibers it innervates

muscle tension
force generated by the contraction of the muscle; tension generated during isotonic contractions and isometric contractions

muscle tone
low levels of muscle contraction that occur when a muscle is not producing movement

myogram
instrument used to measure twitch tension

recruitment
increase in the number of motor units involved in contraction

relaxation phase
period after twitch contraction when tension decreases

tetanus
a continuous fused contraction

treppe
stepwise increase in contraction tension

twitch
single contraction produced by one action potential

wave summation
addition of successive neural stimuli to produce greater contraction

Muscular System Module 5: Exercise and Muscle Performance
By the end of this section, you will be able to:

- Describe hypertrophy and atrophy
- Explain how resistance exercise builds muscle
- Explain how performance-enhancing substances affect muscle

Muscular System Module 5: Exercise and Muscle Performance

Muscular System Module 5: Exercise and Muscle Performance

Physical training alters the appearance of skeletal muscles and can produce changes in muscle performance. Conversely, a lack of use can result in decreased performance and muscle appearance. Although muscle cells can change in size, new cells are not formed when muscles grow. Instead, structural proteins are added to muscle fibers in a process called **hypertrophy**, so cell diameter increases. The reverse, when structural proteins are lost and muscle mass decreases, is called **atrophy**. Age-related muscle atrophy is called **sarcopenia**. Cellular components of muscles can also undergo changes in response to changes in muscle use.

Endurance Exercise

Slow twitch fibers are predominantly used in endurance exercises that require little force but involve numerous repetitions. The aerobic metabolism used by slow-twitch fibers allows them to maintain contractions over long periods. Endurance training modifies these slow fibers to make them even more efficient by producing more mitochondria to enable more aerobic metabolism and more ATP production. Endurance exercise can also increase the amount of myoglobin in a cell, as increased aerobic respiration increases the need for oxygen. **Myoglobin** is found in the sarcoplasm and acts as an oxygen storage supply for the mitochondria.

The training can trigger the formation of more extensive capillary networks around the fiber, a process called **angiogenesis**, to supply oxygen and remove metabolic waste. To allow these capillary networks to supply the deep portions of the muscle, muscle mass does not greatly increase in order to maintain a smaller area for the diffusion of nutrients and gases. All of these cellular changes result in the ability to sustain low levels of muscle contractions for greater periods without fatiguing.

The proportion of slow muscle fibers in muscle determines the suitability of that muscle for endurance, and may benefit those participating in endurance activities. Postural muscles have a large number of slow fibers and relatively few fast fibers, to keep the back straight ([link]). Endurance athletes, like marathon-runners also would benefit from a larger proportion of slow fibers. b

Marathoners

Long-distance runners have a large number of SO fibers and relatively few FO and FG fibers. (credit: “Tseo2”/Wikimedia Commons)

Resistance Exercise

Resistance exercises, as opposed to endurance exercise, require large amounts of **fast twitch fibers** to produce short, powerful movements that are not repeated over long periods. The high rates of ATP use and cross-bridge formation in fast fibers result in powerful muscle contractions.

Muscles used for power have a higher ratio of fast to slow fibers, and trained athletes possess even higher levels of fast fibers in their muscles. Resistance exercise affects muscles by increasing the formation of myofibrils, thereby increasing the thickness of muscle fibers. This added structure causes hypertrophy, or the enlargement of muscles, exemplified by the large skeletal muscles seen in body builders and other athletes ([link]). Because this muscular enlargement is achieved by the addition of structural proteins, athletes trying to build muscle mass often ingest large amounts of protein.

Hypertrophy

Body builders have a large number of FG fibers and relatively few FO and SO fibers. (credit: Lin Mei/flickr)

Except for the hypertrophy that follows an increase in the number of sarcomeres and myofibrils in a skeletal muscle, the cellular changes observed during endurance training do not usually occur with resistance training. There is usually no significant increase in mitochondria or capillary density. However, resistance training does increase the development of connective tissue, which adds to the overall mass of the muscle and helps to contain muscles as they produce increasingly powerful contractions. Tendons also become stronger to prevent tendon damage, as the force produced by muscles is transferred to tendons that attach the muscle to bone.

For effective strength training, the intensity of the exercise must continually be increased. For instance, continued weight lifting without increasing the weight of the load does not increase muscle size. To produce ever-greater results, the weights lifted must become increasingly heavier, making it more difficult for muscles to move the load. The muscle then adapts to this heavier load, and an even heavier load must be used if even greater muscle mass is desired.

If done improperly, resistance training can lead to overuse injuries of the muscle, tendon, or bone. These injuries can occur if the load is too heavy or if the muscles are not given sufficient time between workouts to recover or if joints are not aligned properly during the exercises. Cellular damage to muscle fibers that occurs after intense exercise includes damage to the sarcolemma and myofibrils. This muscle damage contributes to the feeling of soreness after strenuous exercise, but muscles gain mass as this damage is repaired, and additional structural proteins are added to replace the damaged ones. Overworking skeletal muscles can also lead to tendon damage and even skeletal damage if the load is too great for the muscles to bear.

Performance-Enhancing Substances

Some athletes attempt to boost their performance by using various agents that may enhance muscle performance. **Anabolic steroids** are one of the more widely known agents used to boost muscle mass and increase power output. Anabolic steroids are a form of testosterone, a male sex hormone that stimulates muscle formation, leading to increased muscle mass.

Endurance athletes may also try to boost the availability of oxygen to muscles to increase aerobic respiration by using substances such as erythropoietin (EPO), a hormone normally produced in the kidneys, which triggers the production of red blood cells. The extra oxygen carried by these blood cells can then be used by muscles for aerobic respiration. **Human growth hormone (hGH)** is another supplement, and although it can facilitate building muscle mass, its main role is to promote the healing of muscle and other tissues after strenuous exercise. Increased hGH may allow for faster recovery after muscle damage, reducing the rest required after exercise, and allowing for more sustained high-level performance.

Although performance-enhancing substances often do improve performance, most are banned by governing bodies in sports and are illegal for nonmedical purposes. Their use to enhance performance raises ethical issues of cheating because they give users an unfair advantage over nonusers. A greater concern, however, is that their use carries serious health risks. The side effects of these substances are often significant, nonreversible, and in some cases fatal. The physiological strain caused by these substances is often greater than what the body can handle, leading to effects that are unpredictable and dangerous. Anabolic steroid use has been linked to infertility, aggressive behavior, cardiovascular disease, and brain cancer.

Similarly, some athletes have used creatine to increase power output. **Creatine phosphate** provides quick bursts of ATP to muscles in the initial stages of contraction. Increasing the amount of creatine available to cells is thought to produce more ATP and therefore increase explosive power output, although its effectiveness as a supplement has been questioned.

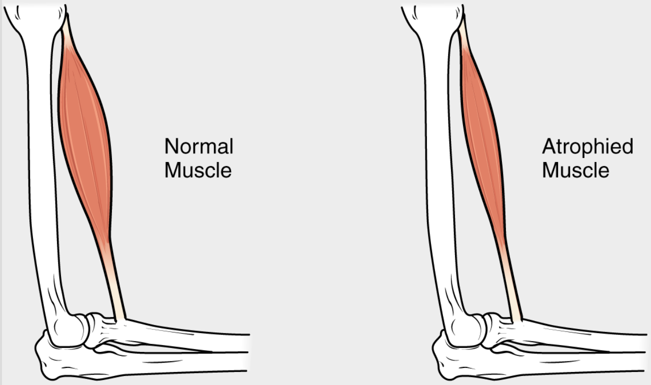
Note:

Everyday Connection

Aging and Muscle Tissue

Although atrophy due to disuse can often be reversed with exercise, muscle atrophy with age, referred to as sarcopenia, is irreversible. This is a primary reason why even highly trained athletes succumb to declining performance with age. This decline is noticeable in athletes whose sports require strength and powerful movements, such as sprinting, whereas the effects of age are less noticeable in endurance athletes such as marathon runners or long-distance cyclists. As muscles age, muscle fibers die, and they are replaced by connective tissue and adipose tissue ([link]). Because those tissues cannot contract and generate force as muscle can, muscles lose the ability to produce powerful contractions. The decline in muscle mass causes a loss of strength, including the strength required for posture and mobility. This may be caused by a reduction in FG fibers that hydrolyze ATP quickly to produce short, powerful contractions. Muscles in older people sometimes possess greater numbers of SO fibers, which are responsible for longer contractions and do not produce powerful movements. There may also be a reduction in the size of motor units, resulting in fewer fibers being stimulated and less muscle tension being produced.

Atrophy



Muscle mass is reduced as muscles atrophy with disuse.

Sarcopenia can be delayed to some extent by exercise, as training adds structural proteins and causes cellular changes that can offset the effects of atrophy. Increased exercise can produce greater numbers of cellular mitochondria, increase capillary density, and increase the mass and strength of connective tissue. The effects of age-related atrophy are especially pronounced in people who are sedentary, as the loss of muscle cells is displayed as functional impairments such as trouble with locomotion, balance, and posture. This can lead to a decrease in quality of life and medical problems, such as joint problems because the muscles that stabilize bones and joints are weakened. Problems with locomotion and balance can also cause various injuries due to falls.

Chapter Review

Hypertrophy is an increase in muscle mass due to the addition of structural proteins. The opposite of hypertrophy is atrophy, the loss of muscle mass due to the breakdown of structural proteins. Endurance exercise causes an increase in cellular mitochondria, myoglobin, and capillary networks in SO fibers. Endurance athletes have a high level of SO fibers relative to the other fiber types. Resistance exercise causes hypertrophy. Power-producing muscles have a higher number of FG fibers than of slow fibers. Strenuous exercise causes muscle cell damage that requires time to heal. Some athletes use performance-enhancing substances to enhance muscle performance. Muscle atrophy due to age is called sarcopenia and occurs as muscle fibers die and are replaced by connective and adipose tissue.

Glossary

angiogenesis
formation of blood capillary networks

atrophy
loss of structural proteins from muscle fibers

hypertrophy
addition of structural proteins to muscle fibers

sarcopenia
age-related muscle atrophy

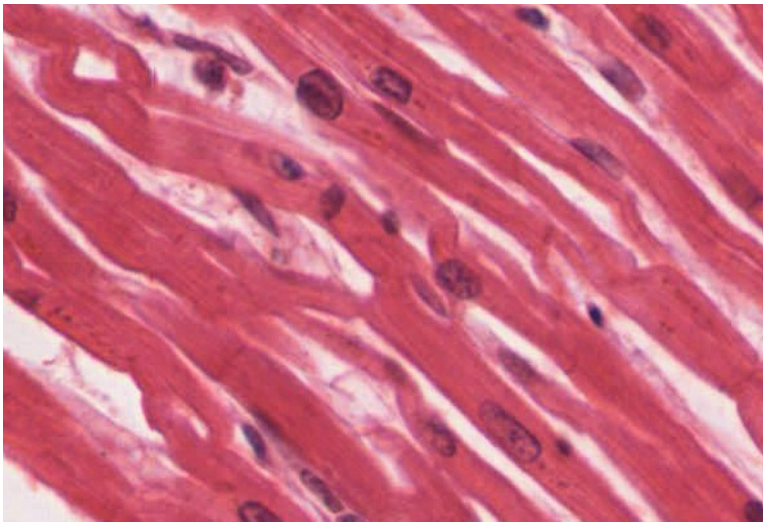
Muscular System Module 6: Cardiac Muscle Tissue

By the end of this section, you will be able to:

- Describe intercalated discs and gap junctions
- Describe a desmosome

Cardiac muscle tissue is only found in the heart. Highly coordinated contractions of cardiac muscle pump blood into the vessels of the circulatory system. Similar to skeletal muscle, cardiac muscle is striated and organized into sarcomeres, possessing the same banding organization as skeletal muscle ([link]). However, cardiac muscle fibers are shorter than skeletal muscle fibers and usually contain only one nucleus, which is located in the central region of the cell. Cardiac muscle fibers also possess many mitochondria and myoglobin, as ATP is produced primarily through aerobic metabolism. Cardiac muscle fibers cells also are extensively branched and are connected to one another at their ends by intercalated discs. An **intercalated disc** allows the cardiac muscle cells to contract in a wave-like pattern so that the heart can work as a pump.

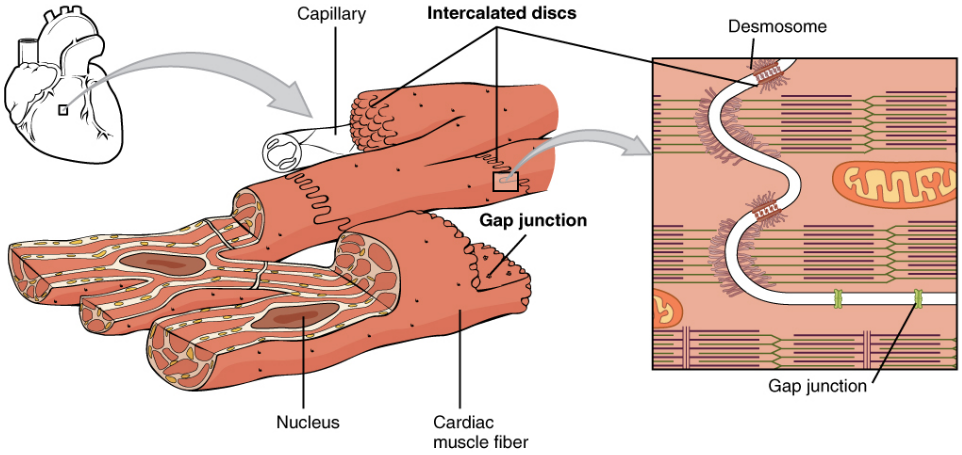
Cardiac Muscle Tissue



Cardiac muscle tissue is only found in the heart. LM × 1600. (Micrograph provided by the Regents of University of Michigan Medical School © 2012)

Intercalated discs are part of the sarcolemma and contain two structures important in cardiac muscle contraction: gap junctions and desmosomes. The **gap junctions** enhance electrical communication between muscle cells, so that all contract at the same time. This network of electrically connected cardiac muscle cells allow all of the muscle cells to contract at the same moment. This creates a functional unit of contraction called a **functional syncytium**. The remainder of the intercalated disc is composed of desmosomes. A **desmosome** is a cell structure that anchors the ends of cardiac muscle fibers together so the cells do not pull apart during the stress of individual fibers contracting ([link]).

Cardiac Muscle



Intercalated discs are part of the cardiac muscle sarcolemma and they contain gap junctions and desmosomes.

Contractions of the heart (heartbeats) are controlled by specialized cardiac muscle cells called **pacemaker cells** that directly control heart rate. Although cardiac muscle cannot be consciously controlled, the pacemaker cells respond to signals from the autonomic nervous system (ANS) to speed up or slow down the heart rate. The pacemaker cells can also respond to various hormones that modulate heart rate to control blood pressure.

The wave of contraction that allows the heart to work as a unit, called a functional syncytium, begins with the pacemaker cells. This group of cells is self-excitable and able to stimulate the rest of the heart to contract, a feature called **autorhythmicity**; they do this at set intervals which determine heart rate. Because they are connected with gap junctions to surrounding muscle fibers and the specialized fibers of the heart’s conduction system, the pacemaker cells are able to transfer the signal to contract to the other cardiac muscle fibers in a manner that allows the heart to contract in a coordinated manner.

Glossary

autorhythmicity
heart’s ability to control its own contractions

desmosome
cell structure that anchors the ends of cardiac muscle fibers to allow contraction to occur

intercalated disc
part of the sarcolemma that connects cardiac tissue, and contains gap junctions and desmosomes

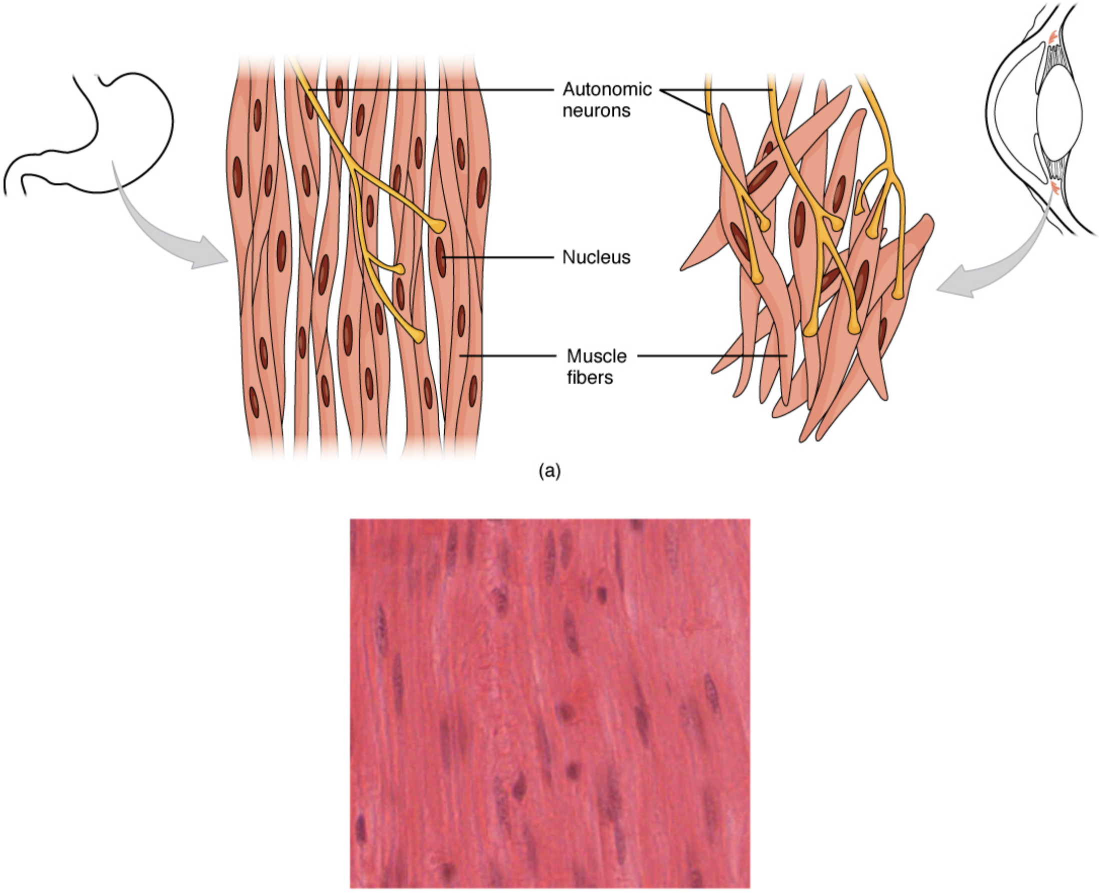
Muscular System Module 7: Smooth Muscle

By the end of this section, you will be able to:

- Describe a dense body
- Explain how smooth muscle works with internal organs and passageways through the body
- Explain how smooth muscles differ from skeletal and cardiac muscles
- Explain the difference between single-unit and multi-unit smooth muscle

Smooth muscle (so-named because the cells do not have striations) is present in the walls of hollow organs like the urinary bladder, uterus, stomach, intestines, and in the walls of passageways, such as the arteries and veins of the circulatory system, and the tracts of the respiratory, urinary, and reproductive systems ([link]**ab**). Smooth muscle is also present in the eyes, where it functions to change the size of the iris and alter the shape of the lens; and in the skin where it causes hair to stand erect in response to cold temperature or fear.

Smooth Muscle Tissue



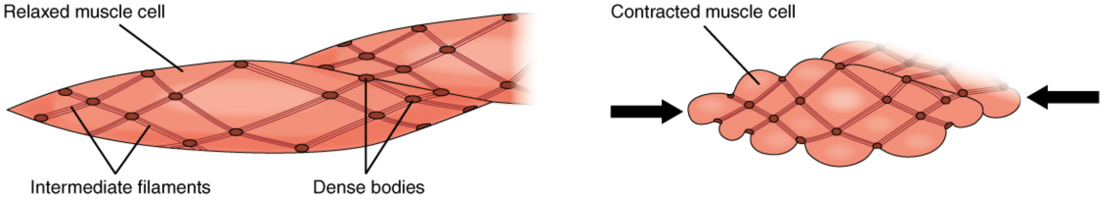
(a)

(b)

Smooth muscle tissue is found around organs in the digestive, respiratory, reproductive tracts and the iris of the eye. LM × 1600. (Micrograph provided by the Regents of University of Michigan Medical School © 2012)

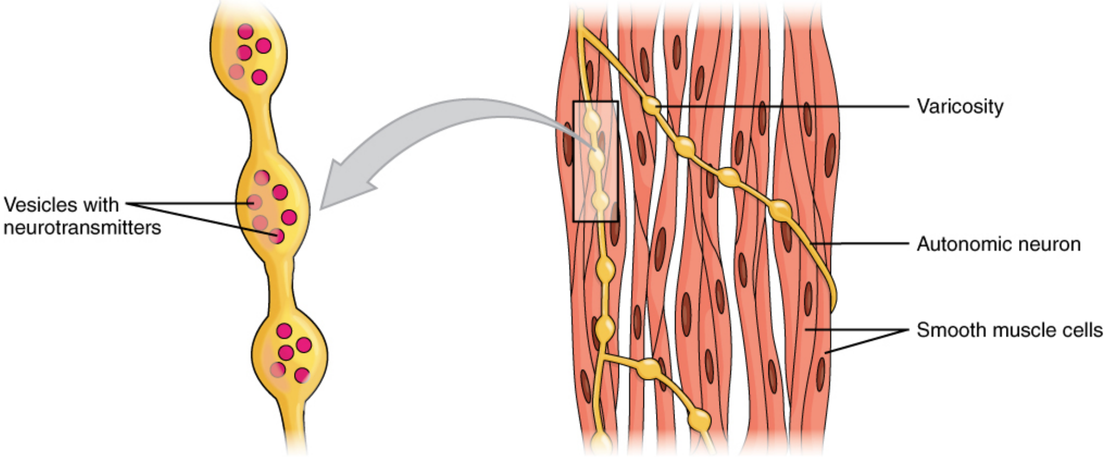
Smooth muscle fibers are spindle-shaped (wide in the middle and tapered at both ends, somewhat like a football) and have a single nucleus. They shorter than skeletal muscle fibers. Although they do not have striations and sarcomeres, smooth muscle fibers do have actin and myosin contractile proteins, and thick and thin filaments. These thin filaments are anchored by dense bodies. A **dense body** functions like a Z-discs of skeletal and cardiac muscle fibers. Because most smooth muscles must function for long periods without rest, their power output is relatively low, but contractions can continue without using large amounts of energy.

Muscle Contraction



The dense bodies and intermediate filaments are networked through the sarcoplasm, which cause the muscle fiber to contract.

Motor Units



A series of axon-like swelling, called varicosities or "boutons," from autonomic neurons form motor units through the smooth muscle.

Glossary

dense body
: sarcoplasmic structure that attaches to the sarcolemma and shortens the muscle as thin filaments slide past thick filaments

visceral muscle

smooth muscle found in the walls of visceral organs

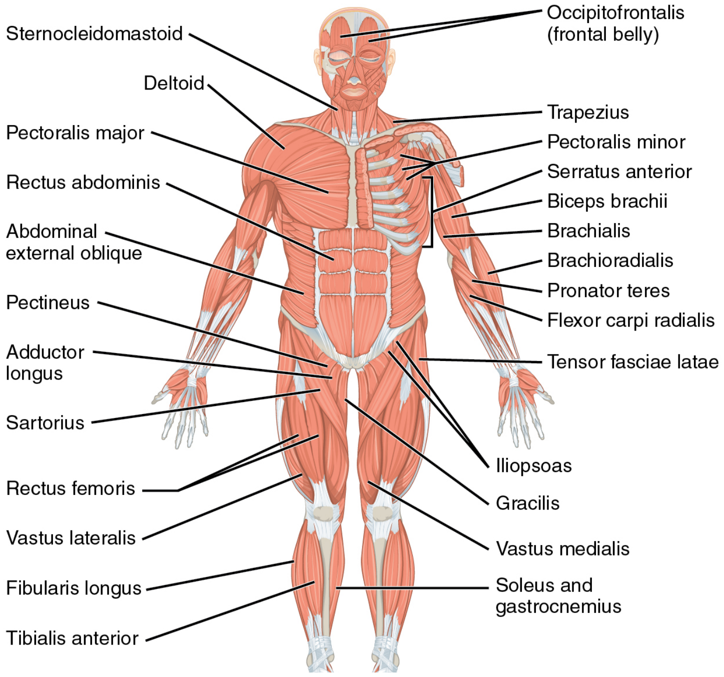
Muscular System Module 8: Gross Anatomy of the Muscular System

By the end of this section, you will be able to:

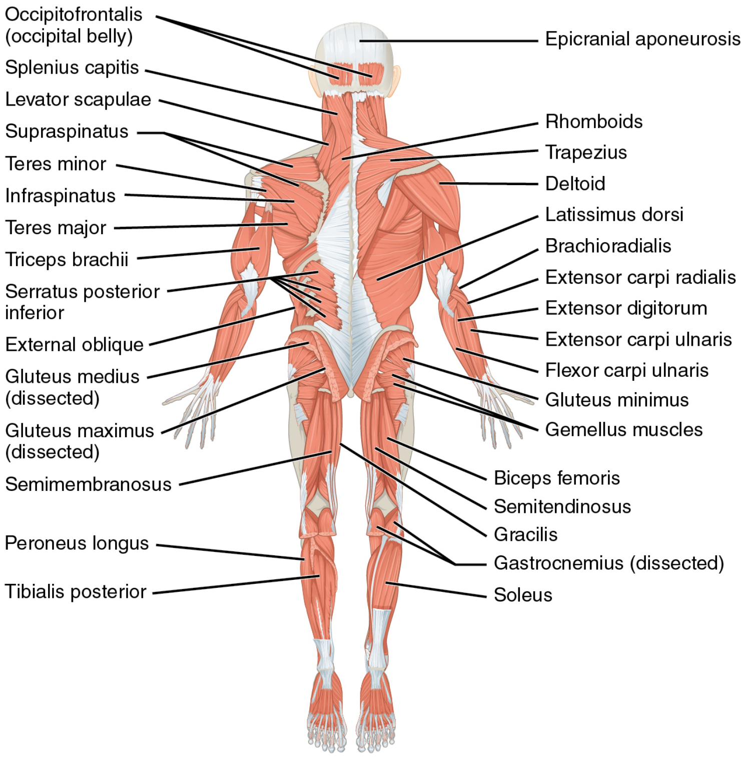
- Describe the criteria used to name skeletal muscles
- Explain how understanding the muscle names helps describe shapes, location, and actions of various muscles

The Greeks and Romans conducted the first studies done on the human body in Western culture. The educated class of subsequent societies studied Latin and Greek, and therefore the early pioneers of anatomy continued to apply Latin and Greek terminology or roots when they named the skeletal muscles. The large number of muscles in the body and unfamiliar words can make learning the names of the muscles in the body seem daunting, but understanding the origin of the names can help. **Etymology** is the study of how the root of a particular word entered a language and how the use of the word evolved over time. Taking the time to learn the root of the words is crucial to understanding the vocabulary of anatomy and physiology. When you understand the names of muscles it will help you remember where the muscles are located and what they do ([link], [link], and [link]). Pronunciation of words and terms will take a bit of time to master, but after you have some basic information; the correct names and pronunciations will become easier.

Overview of the Muscular System



Major muscles of the body.
Right side: superficial; left side:
deep (anterior view)



Major muscles of the body.
Right side: superficial; left side:
deep (posterior view)

On the anterior and posterior views of the muscular system above, superficial muscles (those at the surface) are shown on the right side of the body while deep muscles (those underneath the superficial muscles) are shown on the left half of the body. For the legs, superficial muscles are shown in the anterior view while the posterior view shows both superficial and deep muscles.

Understanding a Muscle Name from the Latin

Example	Word	Latin Root 1	Latin Root 2	Meaning	Translation
abductor digiti minimi	abductor	ab = away from	duct = to move	a muscle that moves away from	A muscle that moves the little finger or toe away
	digiti	digitus = digit		refers to a finger or toe	
	minimi	minimus = mini, tiny		little	
adductor digiti minimi	adductor	ad = to, toward	duct = to move	a muscle that moves towards	A muscle that moves the little finger or toe toward
	digiti	digitus = digit		refers to a finger or toe	
	minimi	minimus = mini, tiny		little	

Mnemonic Device for Latin Roots		
Example	**Latin or Greek Translation**	**Mnemonic Device**
ad	to; toward	ADvance toward your goal
ab	away from	n/a
sub	under	SUBmarines move under water.
ductor	something that moves	A conDUCTOR makes a train move.
anti	against	If you are antisocial, you are against engaging in social activities.
epi	on top of	n/a
apo	to the side of	n/a

Mnemonic Device for Latin Roots		
Example	**Latin or Greek Translation**	**Mnemonic Device**
longissimus	longest	“Longissimus” is longer than the word “long.”
longus	long	long
brevis	short	brief
maximus	large	max
medius	medium	“Medius” and “medium” both begin with “med.”
minimus	tiny; little	mini
rectus	straight	To RECTify a situation is to straighten it out.
multi	many	If something is MULTIcolored, it has many colors.
uni	one	A UNIcorn has one horn.
bi/di	two	If a ring is DIcast, it is made of two metals.
tri	three	TRIple the amount of money is three times as much.
quad	four	QUADruplets are four children born at one birth.

Mnemonic Device for Latin Roots		
Example	**Latin or Greek Translation**	**Mnemonic Device**
externus	outside	EXternal
internus	inside	INternal

Anatomists name the skeletal muscles according to a number of criteria, each of which describes the muscle in some way. These include naming the muscle after its shape, its size compared to other muscles in the area, its location in the body or the location of its attachments to the skeleton, how many origins it has, or its action.

The skeletal muscle’s anatomical location or its relationship to a particular bone often determines its name. For example, the frontalis muscle is located on top of the frontal bone of the skull. Similarly, the shapes of some muscles are very distinctive and the names, such as orbicularis, reflect the shape. For the buttocks, the size of the muscles influences the names: gluteus **maximus** (largest), gluteus **medius** (medium), and the gluteus **minimus** (smallest). Names were given to indicate length—**brevis** (short), **longus** (long)—and to identify position relative to the midline: **lateralis** (to the outside away from the midline), and **medialis** (toward the midline). The direction of the muscle fibers and fascicles are used to describe muscles relative to the midline, such as the **rectus** (straight) abdominis, or the **oblique** (at an angle) muscles of the abdomen.

Some muscle names indicate the number of muscles in a group. One example of this is the quadriceps, a group of four muscles located on the anterior (front) thigh. Other muscle names can provide information as to how many origins a particular muscle has, such as the biceps brachii. The prefix **bi** indicates that the muscle has two origins and **tri** indicates three origins.

The location of a muscle’s attachment can also appear in its name. When the name of a muscle is based on the attachments, the origin is always named first. For instance, the sternocleidomastoid muscle of the neck has a dual origin on the sternum (sterno) and clavicle (cleido), and it inserts on the mastoid process of the temporal bone. The last feature by which to name a muscle is its action. When muscles are named for the movement they produce, one can find action words in their name. Some examples are **flexor** (decreases the angle at the joint), **extensor** (increases the angle at the joint), **abductor** (moves the bone away from the midline), or **adductor** (moves the bone toward the midline).

Muscle Movement

To move the skeleton, the tension created by the contraction of the fibers in most skeletal muscles is transferred to the **tendons**. The tendons are strong bands of dense, regular connective tissue that connect muscles to bones. The bone connection is why this muscle tissue is called skeletal muscle.

To pull on a bone, that is, to change the angle at its synovial joint, which essentially moves the skeleton, a skeletal muscle must also be attached to a fixed part of the skeleton. The moveable end of the muscle that attaches to the bone being pulled is called the muscle’s **insertion**, and the end of the muscle attached to a fixed (stabilized) bone is called the **origin**.

Although a number of muscles may be involved in an action, the principal muscle involved is called the **prime mover**, or **agonist**. To lift a cup, a muscle called the biceps brachii is actually the prime mover; however, because it can be assisted by the brachialis, the brachialis is called a **synergist** in this action ([link]). A synergist can also be a **fixator** that stabilizes the bone that is the attachment for the prime mover’s origin.

A muscle with the opposite action of the prime mover is called an **antagonist**. Antagonists play two important roles in muscle function: (1) they maintain body or limb position, such as holding the arm out or standing erect; and (2) they control rapid movement, as in shadow boxing without landing a punch or the ability to check the motion of a limb.

Skeletal muscle is enclosed in connective tissue scaffolding at three levels. Each muscle fiber (cell) is covered by endomysium and the entire muscle is covered by epimysium. When a group of muscle fibers is “bundled” as a unit within the whole muscle by an additional covering of a connective tissue called perimysium, that bundled group of muscle fibers is called a **fascicle**. Fascicle arrangement by perimysia is correlated to the force generated by a muscle; it also affects the range of motion of the muscle. Based on the patterns of fascicle arrangement, skeletal muscles can be classified in several ways.

The Interaction of the Skeletal and Muscular Systems

Skeletal muscles do not work by themselves. Muscles are arranged in pairs based on their functions. For muscles attached to the bones of the skeleton, the connection determines the force, speed, and range of movement. These characteristics depend on each other and can explain the general organization of the muscular and skeletal systems.

The skeleton and muscles act together to move the body. Have you ever used the back of a hammer to remove a nail from wood? The handle acts as a **lever** and the head of the hammer acts as a **fulcrum**, the fixed point that the force is applied to when you pull back or push down on the handle. The effort applied to this system is the pulling or pushing on the handle to remove the nail, which is the **load**, or “resistance” to the movement of the handle in the system. Our musculoskeletal system works in a similar manner, with bones being stiff levers and the articular endings of the bones —encased in synovial joints—acting as fulcrums. The load would be an object being lifted or any resistance to a movement (your head is a load when you are lifting it), and the effort, or applied force, comes from contracting skeletal muscle.

Glossary

abductor
    moves the bone away from the midline

adductor
    moves the bone toward the midline

bi	two
brevis	short
extensor	muscle that increases the angle at the joint
flexor	muscle that decreases the angle at the joint
lateralis	to the outside
longus	long
maximus	largest
medialis	to the inside
medius	medium
minimus	smallest
oblique	at an angle
rectus	straight
tri	three